

AMENDMENTS TO THE CLAIMS

This listing of claims will replace all prior versions and listings of claims in the application. Additions are shown as underlined and deletions are shown as ~~struckthrough~~.

1. (Currently Amended) A method of treating ~~or preventing~~ a disease involving cell hyperproliferation, comprising ~~inhibiting the interaction between Hecl protein and at least one further protein by administering to a subject a compound comprising a core N-(4-phenylthiazol-2-yl)benzamide structure~~ ~~small molecule drug~~, thereby lessening cell hyperproliferation.
2. (Canceled)
3. (Canceled)
4. (Canceled)
5. (Canceled)
6. (Canceled)
7. (Canceled)
8. (Canceled)
9. (Canceled)
10. (Canceled)
11. (Previously Presented) The method of claim 1, wherein the disease involving cell hyperproliferation is a cancer.

12. (Original) The method of claim 11, wherein the cancer is a carcinoma.
13. (Previously Presented) The method of claim 12, wherein the carcinoma is a carcinoma selected from the group consisting of: bladder carcinoma, breast carcinoma, cervical carcinoma, hepatocellular carcinoma, and prostate carcinoma.
14. (Original) The method of claim 11, wherein the cancer is a sarcoma.
15. (Original) The method of claim 14, wherein the sarcoma is a sarcoma selected from the group consisting of alveolar soft part sarcoma, ameloblastic sarcoma, botryoid sarcoma, clear cell sarcoma of kidney, endometrial stromal sarcoma, Ewing's sarcoma, giant cell sarcoma, hemangioendothelial sarcoma, immunoblastic sarcoma of B cells, immunoblastic sarcoma of T cells, Kaposi's sarcoma, Kupffer cell sarcoma, osteogenic sarcoma, pseudo-Kaposi sarcoma, reticulum cell sarcoma, Rous sarcoma, soft tissue sarcoma and spindle cell sarcoma.
16. (Original) The method of claim 11, wherein the cancer is retinoblastoma, glioblastoma, or neuroblastoma.
17. (Canceled)
18. (Canceled)
19. (Canceled)
20. (Canceled)
21. (Currently Amended) A method of identifying a compound that reduces an interaction between Hec1 protein and Hint1 protein ~~at least one further protein~~, comprising:

- a) contacting Hec1 protein with Hint1 protein and at least one further protein in the relative absence of the compound;
- b) contacting Hec1 protein with Hint1 protein and at least one further protein in the relative presence of the compound; and
- c) determining the relative amount of interaction between the Hec1 protein and the Hint1 protein at least one further protein in a) and b); and
- d) comparing the relative amount of interaction, wherein if the relative presence of the compound causes less interaction than the relative absence of the compound, the compound is identified as a compound that reduces an interaction between the Hec1 protein and Hint1 protein at least one further protein.

22. (Currently Amended) The method of claim 21, wherein the at least one further protein is further comprising contacting Hec1 with Nek2 protein.

23. (Canceled)

24. (Currently Amended) The method of claim 21, wherein the Hec1 protein is immobilized and the relative amount of interaction is determined by measurement of co-immobilization of the Hint1 protein at least one further protein.

25. (Currently Amended) The method of claim 21, wherein the Hint1 protein at least one of the further proteins is immobilized and the relative amount of interaction is determined by measurement of co-immobilization of the Hec1 protein.

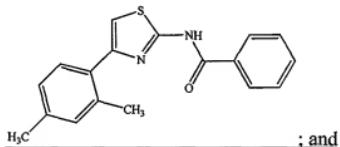
26. (Original) The method of claim 21, wherein b) and c) include immunoprecipitation of proteins.

27. (Currently Amended) The method of claim 21, wherein b) and c) include co-localization of labels specific for Hec1 protein and Hin1 protein the further protein.

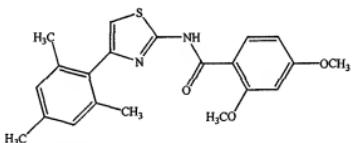
28. (Currently Amended) A method of identifying a molecule that interferes with a function of Hec1 protein, Nek2 protein and/or Hint1protein and inhibits cell proliferation, comprising:
 - a) contacting a sample comprising cells with the molecule or a combination of molecules; and
 - b) measuring the amount that the molecules or combination of molecules interferes with a function of Hec1 protein, Nek2 protein and/or Hint1protein involved in cell proliferation, cell cycle progression, cell cycle arrest, or apoptosis in the sample exposed to the molecule or combination of molecules, whereby a decrease in cell proliferation, a decrease in cell cycle progression, an increase in cell cycle arrest, or an increase in apoptosis in the sample comprising proliferating cells exposed to the molecule or combination of molecules, relative to the amount of proliferation, cell cycle progression, cell cycle arrest, or apoptosis in a sample comprising proliferating cells not contacted with the molecule or combination of molecules, identifies a molecule or combination of molecules that inhibits proliferation of the cells.
29. (Original) The method of claim 28, wherein the sample comprises isolated cells.
30. (Original) The method of claim 28, wherein the sample a tissue sample.
31. (Original) The method of claim 28, wherein the sample is a tissue sample in an organism.
32. (Canceled)
33. (Canceled)
34. (Canceled)
35. (Canceled)

36. (Canceled)

37. (Currently Amended) The method molecule of claim 1 claim 36, wherein the compound molecule is selected from the group consisting of N-[4-(2,4-dimethylphenyl)thiazol-2-yl]benzamine, IBT13131, having the formula:



N-[4-(2,4,6-trimethylphenyl)thiazol-2-yl]-2,4-dimethoxybenzamine, IBT14664, having the formula:



38. (Currently Amended) A composition comprising a compound identified by the method of Claim 21, that comprises a core N-(4-phenylthiazol-2-yl)benzamide structure molecule or ligand of claim 36 and a pharmaceutically acceptable carrier.

39. (Currently Amended) The method of claim 1, wherein the core N-(4-phenylthiazol-2-yl)benzamide structure has additional groups on the benzene rings small-molecule drug contains a core phenyl-thiazol-benzamide structure.

40. (Canceled)

41. (Canceled)
42. (Previously Presented) The method of claim 11, wherein the cancer is an Rb-deficient cancer.